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CASE REPORTS

Hiccup and Heart Block

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Transient heart block may be induced by vagal stimulation, and it has been described as a complication of a number of physiological acts involving vagal activity, such as deep breathing (Schwartz and Schwartz, 1961), "gagging" (Dressler, 1959), and swallowing (Weiss and Ferris, 1934; Deuchar and Trounce, 1960), as well as exertion (Gough and Galpin, 1964); though with swallowing there is probably an underlying abnormality of the pharynx such as glosso-pharyngeal neuralgia, or of the oesophagus, such as a pouch. The writer has been unable to find any report of the association of heart block with hiccup and therefore presents the following case history.

Case History

An 87-year-old man, already in hospital because of prostatic obstruction, became pulseless and unconscious while defaecating after an enema. He recovered after about a minute, and when seen an hour later was fully conscious and hiccupping. The pulse was regular at 76/min., but occasional transient slowing of the rate was noted. The blood pressure was 170/90 mm. Hg. There was grade 3/6 aortic ejection systolic murmur, and a heaving apex beat. An electrocardiogram demonstrated nodal rhythm at 76/min. There were, in addition, a number of wave forms occurring at irregular intervals, both singly and in groups, each wave coinciding with a hiccup. Single hiccups for the most part had no influence on the underlying cardiac rhythm; but a quick succession of hiccups was usually followed by nodal arrest, succeeded by idioventricular rhythm at 37/min. apparently arising from a focus in the left ventricle (Fig. 1). This lasted for up to 6 cycles and was followed by return to nodal rhythm. The ventricular complexes themselves demonstrated sagging of S–T segments in standard and left ventricular leads together with prolongation of QTc to about 0·4 sec. There was a past history of atrial fibrillation and congestive heart failure, and the patient was taking digoxin 0·25 mg. twice daily and chlorothiazide 1 g. on alternate days. He was taking no potassium supplements. The serum electrolytes were as follows: sodium 129 mEq/litre, potassium 1·9 mEq/litre, chlorides 82 mEq/litre, bicarbonates 32 mEq/litre, urea 30 mg./100 ml.

Chlorothiazide and digoxin were stopped and potassium was started, 3 g. potassium chloride being given intravenously and 7·2 g. orally in the first 24 hours, as well as chlorpromazine 50 mg. intramuscularly 6-hourly.

By the next day the patient's general condition had improved. He was still hiccupping but not so often; the hiccups still induced changes from nodal to idioventricular rhythm, however (Fig. 1b). Now the ventricular beats appeared to originate from the right ventricle and the rate was faster at 53/min. against a current nodal rate of 58/min. For this reason changes in rhythm were not clinically observable. The electrocardiograms showed sagging of S–T segments and also T-U complexes suggestive of hypokalaemia. The serum electrolyte figures were as follows: sodium 132 mEq/litre, potassium 2·7 mEq/litre, chlorides 94 mEq/litre, bicarbonate 35 mEq/litre, urea 27 mg./100 ml. During the next 3 days there was further improvement, the hiccups became less frequent and stopped and the electrolytes returned to normal levels: *30.1.68*—sodium 132 mEq/litre, potassium 3·5 mEq/litre, chloride 94 mEq/litre, bicarbonate 29 mEq/litre, urea 30 mg./100 ml.; *2.2.68*—sodium 132 mEq/litre, potassium 3·9 mEq/litre, chloride 94 mEq/litre, bicarbonate 25 mEq/litre, urea 30 mg./100 ml.; *9.2.68*—sodium 135 mEq/litre, potassium 4·1 mEq/litre, chloride 104 mEq/litre, bicarbonate 25 mEq/litre, urea 37 mg./100 ml.

At this time an electrocardiogram showed atrial fibrillation at about 90/min. The QRS complexes were normal apart from showing flattening of T waves over the standard and unipolar limb leads (Fig. 1c). Prostatectomy was performed a week later (15.2.68), and the patient was discharged to convalesce a month after that (14.3.68). He died suddenly 3 to 4 weeks later, however. There was no necropsy.

Discussion

Hiccup and Heart Block. There have been several reports on the association of hiccup with abnormalities of the heart and of its rhythm. Its occurrence with angina, induced either by exercise

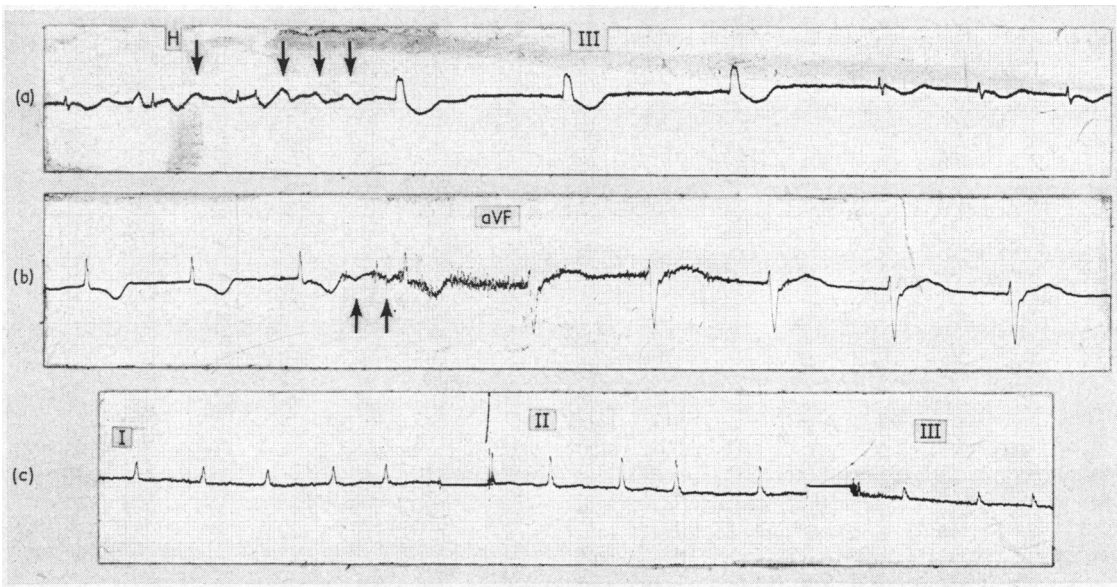

FIG. 1.—(a) Nodal rhythm at 76 to the minute with a series of hiccups (arrows) followed by nodal arrest and then by idioventricular rhythm at 37 to the minute arising from a focus in the left ventricle. (b) On the next day. Nodal rhythm at 58 to the minute interrupted by hiccups (arrows) and followed by idioventricular rhythm derived from a focus in the right ventricle at a rate of 53 to the minute. This figure shows the difference between the "hiccup-waves" and the succeeding high frequency oscillations due to other somatic muscle contractions. (c) 4 days later; and after restoration of serum potassium to normal levels.

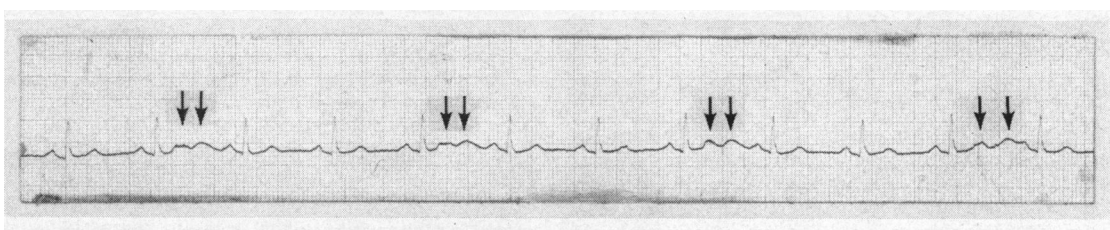

FIG. 2.—Tracing from another subject, without electrolyte imbalance, showing a 3:1 heart beat:hiccup relationship.

or emotion, has been reported (Chavany and Hagenmuller, 1950), as has its association with acute myocardial infarction (Swan and Simoson, 1952; Weiss, 1939; Rubin *et al.*, 1951). The writer has been able to find only one account of hiccup actually inducing an arrhythmia. Cheng and Miller (1953) reported an episode of atrial flutter which started during, and which outlasted a prolonged bout of hiccups, and speculated that "a vagus nerve reflex" was involved. There have, on the other hand, been reports suggesting that the heart beat might in some way initiate hiccups.

Söderström (1950) reported a patient with pyloric stenosis who had alkalosis and tetany and whose left diaphragmatic contractions were synchronous with the heart beat. The hiccups were terminated by correction of the alkalosis. Lepeschkin (1954) described 5 patients in whom hiccups always occurred between 0·17 and 0·4 sec. after the beginning of the QRS complex. He postulated that under certain conditions, such as hypocalcaemia or alkalosis, the phrenic nerve became excited by myocardial action potentials as it ran in approximation to the cardiac muscle. There was no evidence that in his cases the diaphragmatic contractions were confined to the left side, however. Possibly a rhythmic association between hiccup and the heart beat is not uncommon, and the author has recently seen a patient in whom hiccups occurred with every third heart beat (Fig. 2).

In the case here reported, it appeared that the hiccups induced temporary heart block. This was more likely to occur after a short series of hiccups

than after a single one, and it seems probable that the resulting Valsalva situation induced the heart block as a vagal summation effect superimposed on his digitalized hypokalaemic condition.

Heart Block and Hypokalaemia. Hypokalaemia may induce cardiac dysrhythmias including atrial fibrillation, atrial tachycardia, and ectopic beats. Hypokalaemic heart block has also been reported (Guyer, 1964). Conversely, potassium infusions have been found to stop consistently the atrioventricular blocking effect of acetylcholine in dogs, and to correct vagally induced atrioventricular block (Fisch, Feigenbaum, and Bowers, 1963; Feigenbaum, Wunsch, and Fisch, 1965). This patient's heart was already beating in nodal rhythm which is an accepted complication of digitalis therapy; and it is postulated that the underlying hypokalaemia and alkalosis set the stage for the development of episodic hiccup-induced complete heart block.

Hypokalaemia and Hiccup. This association has already been quoted (Söderström, 1950; Lepeschkin, 1954), and hypokalaemia was probably the cause of hiccups here, for they were readily resolved by correction of the patient's hypokalaemic alkalotic state. Hypocalcaemia has also been cited as a cause of hiccups (Corsdress, 1926). In this patient the serum calcium level was not estimated at first; subsequently, however (6.3.68), it was found to be low at 8·6 mg./100 ml., and this may have been a contributory factor, for though calcium was not given therapeutically, correction of the state of alkalosis would have resulted in an increase in the ionized fraction.

Hiccups and the Electrocardiogram. The electrocardiographic complexes caused by hiccups have been discussed by Söderström (1950) who described two typical wave forms; firstly, diphasic waves which he referred to as myograms and which he believed to represent an action potential in the diaphragmatic and synergistic muscles; and secondly, broad upward deflections termed mechanograms, which he considered were due to bodily vibrations arising close to the insertion of leaves of the diaphragm. Lepeschkin (1954), on the other hand, maintained that the action potential in the diaphragm, being tetanic, would result in high frequency oscillations similar to other somatic muscle contractions. He considered that the changes were caused by movements of the body as a whole, which resulted in alterations in tension in the cables leading to the various electrodes. This seems the more likely explanation, and the hiccups complexes of the reported patient accorded with it.

Summary

In an 87-year-old man, hiccups were found to induce transient episodes of nodal arrest with idioventricular rhythm. The patient was hypokalaemic and alkalotic and digitalized. Stoppage of digoxin and correction of the biochemical abnormalities was followed by cessation of hiccups and of the episodic heart block. The possible causes of this association are discussed, and earlier reports relevant to this patient's state of hypokalaemia, hiccup, and heart block are reviewed.

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